POSTDURAL PUNCTURE HEADACHE AND EXTRADURAL BLOOD PATCH

The association of postural headache with puncture of the dura mater was known to the early pioneers of spinal anaesthesia. In 1898, August Bier, the "father" of surgical spinal anaesthesia suffered a severe postural headache after an experimental spinal block performed on himself had to be abandoned when a poor fit between syringe and needle resulted in considerable loss of cerebrospinal fluid (CSF). Nearly 100 years later, the exact pathophysiology of the symptoms is still uncertain, although it is known that loss of spinal CSF depletes the intracranial CSF "cushion" of the brain. Movement of the brain in the upright posture then causes traction on pain-sensitive intracranial structures. Pain arising on or above the tentorium cerebelli is transmitted via the trigeminal nerve to the frontal region; pain from below the tentorium is transmitted via the vagus and upper cervical nerves to the occipital region and neck.

Whatever the exact cause of the pain, the cure is to seal the hole in the dura. This always occurs naturally, usually within 1 week, but it may occasionally take much longer. A remarkable (and unwelcome) feature of the postdural puncture headache (PDPH) syndrome is that such a trivial injury may cause such an incapacitating condition. The postural nature of the symptoms makes them particularly disabling and, as a result, attempts to find a cure have been numerous, unsuccessful and at times ill-advised. Tourtellotte listed alphabetically 49 methods of treatment, from abdominal binders (for which at least there was some physiological basis) to x-radiography of the skull (for which there was not). Nelson attempted the ingenious method of passing a length of catgut down the offending needle to seal the hole, but unfortunately this sometimes resulted in cauda equina lesions [1]. Brown and Jones [2] and Harrington, Tyler and Welch [3], as a last resort (but fortunately successfully), carried out laminectomies to close, with clips or a fascial graft, dural holes which were respectively 5 months and 5 years old! Rather more success—although not invariable—has been claimed for the use of extradural saline boluses [4, 5] or infusion [6], and for i.v. caffeine sodium benzoate [7, 8].

The first report of the use of autologous blood injected into the extradural space to "patch" the hole in the dura was made by Gormley in 1960 [9], inspired by his impression (since shown to be erroneous) that dural puncture accompanied by a "bloody tap" was less likely to result in PDPH. Using only 2–3 ml of blood, he claimed 100% cure in his series of eight subjects (one of whom was himself when suffering from PDPH after myelography). The next significant report was by Odzil and Powell who, using a rather complicated technique of injecting 3 ml of clotted blood into the subarachnoid space and during withdrawal of the needle into the extradural space, claimed no case of PDPH in 100 spinal blocks using a 20-gauge needle, compared with a 15% incidence in a control group [10].

In 1970 [11] and 1972 [12], DiGiovanni and colleagues described two series totalling 108 patients who underwent a blood patch with 5–10 ml of blood with immediate and permanent cure in 90% and relief in another 8% within 24 h. They also performed laboratory experiments in Angora goats showing that injected blood had largely disappeared from the extradural space in 2 weeks. The technique rapidly became more popular, with anaesthetists relieved to be able to offer effective treatment to sufferers from PDPH. The biggest study was a prospective and multi-centre trial organized by the American Society for Obstetric Anesthesia and Perinatology; this consisted of 185 patients, 182 of whom were completely and permanently relieved of PDPH by an average of 10 ml of blood injected 4 days after the onset of symptoms [13].

The success of the method seemed to have been established, but then argument began as to whether or not blood patching should be carried out early, or even prophylactically. Some workers reported very poor success rates with such use [14, 15], while others claimed good results, stating that the essential ingredient for success was to use adequate volumes of blood (15–20 ml) [16–18]. This argument is unresolved, although the possibility that the truth lies somewhere between is suggested by the work of Colonna-Romano and Shapiro, who found that 15 ml of blood given prophylactically reduced the incidence of PDPH from 76.5% to 17.6%—a considerable reduction, but not so spectacularly successful as some reports in which patching was delayed [19].

In the 30 years that blood patching has been performed, there has been conjecture, but very little evidence, on the mode of action and fate of the injected blood. It was assumed that the blood acted as a plug, and exerted a mass effect in the extradural space for an uncertain duration of time during which the body's reparative mechanisms sealed the hole permanently.

DiGiovanni and colleagues had shown that blood clot effectively sealed holes in a plastic bag of saline. Abouleish and colleagues [20], Naulty and Herold...
and others had shown that extradural block was successful when performed after blood patching, suggesting no residual adhesions in the extradural space; and there were no long-term neurological sequelae. Hardy showed, by myelography, that the blood was still present as a space-occupying mass 1 week after injection [22], and Szefinfeld and colleagues [23], in a useful (but unlikely to be repeated) study, used radioactively-labelled red blood cells to show that the injected blood spread extensively, mainly cephalad from the site of injection, to the extent of about 1.6 ml per spinal segment. Cook and Watkins-Pitchford made the interesting laboratory discovery that blood clotting was accelerated in the presence of cerebrospinal fluid [24].

Rosenburg and Heavner gained useful information from in vitro experiments on the ability of extradural blood patches of 25-gauge and 19-gauge holes in dog dura to resist pressures similar to those in the lumbar subarachnoid space of the human in the sitting position [25]. After 30 min of exposure of the dura to autologous blood, the blood was found to be adherent in thin sheets over and around the hole in the dura, with clotted blood protruding in a band through to the cerebrospinal fluid side of the hole in several subjects. The patches were then effective at resisting pressures up to 40 mm Hg in the 25-gauge group, although rather less effective in resisting pressures in the 19-gauge group.

In the current issue of this Journal, an original and informative study of extradural blood patches using magnetic resonance imaging (MRI) pulls together some of these threads of evidence [26]. Imaging was carried out once each in five patients at intervals of 30 min, 3, 7, 9 and 18 h after blood patching. At 30 min and 3 h, the clot was shown to have a mass effect, compressing the dural sac and displacing the conus medullaris and cauda equina. There was also compression or displacement of nerve roots nearby. The main bulk of the clot occupied four or five vertebral levels with thinner spread cephalad and caudad several vertebrae further. Some blood initially increased the CSF signal, indicating its entry into the subarachnoid space, but by 3 h this had concentrated to a focal clot within the dural sac apparently adherent to the dura. Imaging from 7 h onwards showed that the mass effect had disappeared, with a thinning layer of blood adherent to the dural sac, but extending much further cephalad than caudad.

With its images of injected blood initially producing a mass effect resolving to a thinner layer of clot adherent to the posterior aspects of the dural sac, this study confirms earlier assumptions and investigations of the mechanism of extradural blood patch. Temporary displacement of nerve roots corresponds to radicular pain which occasionally accompanies the technique. A surprising feature, not described previously, is the extensive spread of blood back into the subcutaneous fat, and it is suggested that this may be associated with the backache which not infrequently follows blood patching. A tendency for the blood to spread cephalad was noted by Szefinfeld and colleagues [23] and confirms their recommendation that the same or a lower interspace be used for blood patching.

As the study unfolds, it is easy to forget that (for obvious reasons) the five MRI reports are of five different patients. The assumption is made that this is the typical sequence of events which would happen with any individual blood patch and, while probably reasonable, it is still an assumption and for that reason should be confirmed by additional studies. Lastly, while providing useful information on what happens when extradural blood patch is effective, it gives less clue on what happens when it is not.

L. E. S. Carriere
Oxford

REFERENCES
21. Naulty JS, Herold R. Successful epidural anesthesia following